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Viewpoints

An obesity epidemic booga booga?

Currently, one-fifth of Western Europeans have a body mass index (BMI) $\geq 30\,kg/m^2$, a common definition of obesity. On one hand, the increasing prevalence of obesity during the last decades has been reported as a 'major public health issue', a '21st century pandemic', up to an apocalyptic 'obesity tsunami'. Others consider that the trend will induce consequences comparable with global warming. The foreseeable response to these frightening messages has been an upsurge of task forces, committees and agencies that are planning ambitious campaigns against high BMI.

On the other hand, some experts state that the epidemic of obesity is exaggerated,[1] is merely the expression of a subtle shift in the distribution of BMI[2] and, eventually, that there is no epidemic.[2,3] Further, all the noise about obesity and its consequences have fuelled obsessions about body weight in the population—a dieting epidemic—but also confusion on the real consequences of obesity. In response to this 'obesity epidemic booga booga',[4] fat acceptance, anti-dieting and health-at-every-size-movements have gained importance to recall that some excess fat does not mean necessarily bad health and to fight prejudice and discrimination against obese people.

In this point of view, we argue that dramatization of obesity could blur the public health issues at stake. No doubt that mean BMI has increased and that obesity is more frequent than previously. Still, a careful evaluation of the genuine consequences of BMI trends on the health of populations is needed. This requires addressing issues about the nosological status of obesity and its causal role on associated conditions.

Clinical vs population consequences of obesity

Let us first paraphrase one of the Rose's paradigm: 'clinical' (or individual) consequences of overweight and obesity may be distinct from 'population' consequences. In other words, the impact of obesity on population health is likely to differ from its consequences at individual level. The latter are well described, e.g. hypertension, type 2 diabetes, cardiovascular diseases (CVDs), osteoarthritis and several cancers, as well as, probably, depression.

At the population level, the impact of obesity is much more difficult to characterize.[5] While rising diabetes trends, a major threat at population level, have clearly paralleled rising obesity trends in many countries, hypertension or dyslipidaemia have not.[6] For instance, mean blood pressure decreased regularly between the mid-1980s and the mid-1990s in many European and other populations.[7] Moreover, in the USA, the CVD risk associated with obesity is now lower than before, and this is not solely because of the better clinical management of CVD risk factors or the lower prevalence of smoking.[6]

Further, since the early 1980s, coronary heart disease mortality rates have halved and life expectancy has increased steadily in several developed countries.[8] However, obesity has increased in these countries over the same time period. These differing trends are difficult to reconcile with the supposedly potent impact, at the 'population' level, of overweight and obesity on CVD or life expectancy. At the most, the increase in obesity may have slowed down the favourable trends of reduced mortality and increased life expectancy.

In several high-income countries, a levelling off of the previously falling CVD rates was recently observed in young adults;[8] some argued that it is due to obesity trends. However, this might also reflect that most CVD cases in young adults are now sporadic, at least in the wealthier segments of the population: a lower epidemiological plateau may have been reached (floor effect) and further reduction of the low absolute rates of CVD may be not possible. In any case, it is unlikely that the current BMI trends will push the CVD rates back to the high values observed in the 1960s. Obesity is probably not a key player in CVD trends.

Nevertheless, it is clear that obesity does have a large impact on the epidemiology of several important conditions, especially diabetes. A further and often neglected consequence of obesity is chronic disabilities, resulting from conditions strongly associated with obesity such as diabetes and osteoarthritis.[6] Chronic disabilities may also be more severe than before because overweight tends to appear at younger age and lasts for a long time. Potentially, disabilities may be the most substantial public health consequence of obesity, conducing to a decline of healthy ageing and a corresponding increase of the costs of health and social services.

Causality of obesity on associated conditions

Historically, overweight was first found to be a marker of high risk of death in life insurance cohorts, used to determine premiums. Insurers were not interested by knowing whether overweight was causally related or not to any diseases; overweight was merely a risk marker.

How BMI is associated with the risk of chronic disease or mortality is vividly debated. Unlike the monotonic upward (log-linear) risk of mortality associated with many CVD risk factors, there is a U-shaped relationship between BMI and mortality.[9]

While severe obesity (i.e. BMI >35 or $40\,kg/m^2$) is clearly associated with chronic diseases and with a reduced lifespan,[9] slightly elevated BMI may be protective,[10] in particular in elderly. Actually, the 'ideal' BMI is likely to vary across populations, to differ between male and female, to change with age and, maybe, to have changed over time.[10] A consequence for population-based prevention strategies is that shifting the whole BMI distribution towards lower values is not appropriate since low BMI is associated with increased mortality.

Several physiopathological mechanisms, suggesting a causal effect, have linked excess adiposity to a variety of diseases. However, corpulence is a proxy for many variables[2] which may be difficult to tease. The association between obesity and a specific disease (e.g. CVD) may be confounded by numerous factors, socio-economic status being one of them. Potentially, any association between obesity and the disease may be due to factors leading to both rather than being a genuine causal effect of obesity itself.[11] For instance, low physical activity may cause both obesity and

CVD; in this case, obesity is a by-product of low physical activity or, at the most, an intermediate outcome between low physical activity and CVD.

Further, to demonstrate the causal effect of an exposure (obesity) on an outcome (a specific disease), the key is to identify 'interventions' that are able to reduce the exposure (obesity) initially and the outcome (say, CVD) consequentially.[11] Currently, we are short in terms of evidence because we still lack well-defined interventions to modify the mean level and distribution of BMI in populations.

Nosological status of obesity

Another issue is the nosological status of obesity. The call to consider obesity (defined as high BMI) a 'disease' rather than a 'risk factor' is based on a 'utilitarian perspective',[12] the argument being that labelling obesity a disease may potentially help to solicit resources into prevention, treatment and research of obesity, and to reduce stigma and discrimination of obese persons.[12]

However, shifting from the status of obesity as a 'risk factor' to obesity as a 'disease' may not help understand the public health stakes of obesity. The status of risk factor only implies a probabilistic notion for a risk associated with a given BMI, which is undisputable, but does not require any agreement on the essence of obesity or on causal mechanisms linking obesity and related diseases. Critical is the fact that there is no clear-cut difference between obese and non-obese states. The existence of 'healthy obese', i.e. obese with normal cardio-metabolic profile and no co-morbidities, questions the validity of labelling obesity as a disease. Healthy obesity also opens stimulating avenues for a better understanding of physiopathological mechanisms of obesity-associated diseases and for the potential benefit of health promotions in obese persons.

More data and less modelling to prevent the obesity booga booga

Dramatization of overweight and obesity is unlikely to help the public health management of the problem. We suggest that the word 'epidemic' should not to be used to describe rising obesity trends. An epidemic is an occurrence of diseases that is temporarily of high prevalence in a population. By the late 20th century, the definition has been extended to include the increasing prevalence of some risk factors. However, the word epidemic often implies 'an impending danger and a large number of victims'.[13] Still, it is arguable that a large number of victims are caused by overweight and obesity and, more importantly, would all be avoided if the mean BMI was reduced.

The flourishing number of modelling studies is also problematic and may be misleading. For instance, to quantify the potential benefit of obesity prevention, the attributable fraction of high BMI is often computed. For a given disease, the attributable fraction provides the proportion of diseased persons in the population that could be avoided if all the excess risk associated with high BMI was eliminated.[14] Strictly speaking, this presupposes a hypothetical intervention thanks to which the obese people never became obese and were always like non-obese.[14] However, such intervention does not exist and, in fact, the attributable fraction does not accurately inform policy on the disease burden which would be eliminated if high BMI was prevented.[11,14] For policymakers, the key is to get operational interventions to reduce BMI. Different interventions, e.g. diet and physical activity vs drugs and surgery, will not reduce the burden of diseases associated with obesity to the same extent.[11] Obviously, we can not assess with confidence the effect of such interventions on the burden of associated diseases. Modelling helps but is not sufficient to anticipate the consequences of obesity and, above all, to prioritize decisions in health policy with the current level of knowledge.

We argue that the effects of overweight and obesity rising trends still have to be better evaluated at a population level. In European countries, national surveys are needed to assess the distribution of overweight and obesity (using BMI and, naturally, other tools to assess adiposity) and the burden of associated conditions, including chronic disabilities. It is also crucial to evaluate the appropriateness and the impact of pharmacological treatments of risk factors which are prescribed on a massive scale in the obese but subjectively healthy individuals.[15] Dieting habits also need to be better described.

A new international initiative, similar to what was done for MONICA for CVD, could help assess the real nature of the 'epidemic' we are facing. Coupled with the evaluations of prevention strategies, these data would help assess the consequences of obesity at population level and what could be the real benefit of obesity prevention.

References

1 Basham P, Luik J. Is the obesity epidemic exaggerated? Yes. *Br Med J* 2008;336:244.

2 Campos P, Saguy A, Ernsberger P, et al. The epidemiology of overweight and obesity: public health crisis or moral panic? *Int J Epidemiol* 2006;35:55–60.

3 Friedman JM. Obesity: causes and control of excess body fat. *Nature* 2009;459:340–2.

4 Kate H. Shapely Prose. Available at: http://kateharding.net/ (accessed on 20 July 2009).

5 Couzin J. A heavyweight battle over CDC's obesity forecasts. *Science* 2005;308:770–1.

6 Gregg EW, Guralnik JM. Is disability obesity's price of longevity? *JAMA* 2007;298:2066–7.

7 Tunstall-Pedoe H, Connaghan J, Woodward M, et al. Pattern of declining blood pressure across replicate population surveys of the WHO MONICA project, mid-1980s to mid-1990s, and the role of medication. *Br Med J* 2006;332:629–35.

8 Capewell S, O'Flaherty M. What explains declining coronary mortality? Lessons and warnings. *Heart* 2008;94:1105–8.

9 Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* 2009;373:1083–96.

10 Flegal KM, Graubard BI, Williamson DF, Gail MH. Cause-specific excess deaths associated with underweight, overweight, and obesity. *JAMA* 2007;298:2028–37.

11 Hernán MA, Taubman SL. Does obesity shorten life? The importance of well-defined interventions to answer causal questions. *Int J Obes* 2008;32(Suppl 3):S8–14.

12 Allison DB, Downey M, Atkinson RL, et al. Obesity as a disease: a white paper on evidence and arguments commissioned by the Council of the Obesity Society. *Obesity* 2008;16:1161–77.

13 Green MS, Swartz T, Mayshar E, et al. When is an epidemic an epidemic? *Isr Med Assoc J* 2002;4:3–6.

14 Levine BJ. The other causality question: estimating attributable fractions for obesity as a cause of mortality. *Int J Obes* 2008;32(Suppl 3):S4–7.

15 Greene JA. *Prescription by numbers. Drugs and the definition of disease*. Baltimore: The John Hopkins University Press, 2007.

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